57. (Amended) Cut flowers from the transgenic plant of claim 36, comprising the CKI which interacts with CDC2a that was introduced into the parent plant.

REMARKS

In response to the Office Action of April 9, 2002, Applicants have amended the claims which, when considered with the following remarks, is deemed to place the present application in condition for allowance. Favorable consideration of all pending claims is respectfully requested.

In the Office Action of April 9, 2002, the Examiner has made final the restriction requirement issued previously and has withdrawn Claims 1, 3-6, 26, 32-35 and 58-59 from consideration. By this amendment, Applicants have canceled Claims 1, 3-4, 6, 26, 32-35, and 58-59 without prejudice. Applicants reserve the right to file one or more divisional applications directed to the subject matter of the canceled claims.

The specification has been objected to based on a number of different informalities. As requested by the Examiner, the specification has been amended to correct the informalities. Withdrawal of the objection to the specification is respectfully requested.

Claims 2, 5, 7-25, 27-31 and 36-57 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly violative of the written description requirement. It is the Examiner's position that although the specification describes three novel nucleotide sequences (SEQ IDNOs:1, 3, and 5) that encode polypeptides (SEQ ID Nos:2, 4, and 6) "having known homology to cyclin-dependent kinase inhibitors", phenotypic effects are disclosed for plants expressing only one of the disclosed sequences (SEQ ID NO:1 which encodes the polypeptide set forth in SEQ ID NO:2). Thus, the Examiner states on page 5 of the Office Action that in view of the

level of knowledge and skill in the art, one skilled in the art would not recognize from the disclosure that the applicant was in possession of the genus that comprises cyclin-dependent kinases that control or alter growth characteristics."

Under the Written Description Guidelines, *Fed. Reg. Vol. 66,* No. 4, January 5, 2001, pages 1099-1111, the Examiner has the initial burden, after a thorough reading and evaluation of the application, of presenting evidence or reasons why one skilled in the art would not recognize that the written description of the invention provides support for the claims. The Examiner has presented reasons for the written description rejection based on findings presented by Nakayama et al. (1998) *BioEssays 20*:1020-1029. The article by Nakayama et al., teaches *inter alia*, that although the members of the Cip/Kip family are structurally and biochemically similar, the range of phenotypes observed in knockout mice of different Cip/Kip members suggests that different members of the Cip/Kip family may nonetheless have diverse physiological functions. The Examiner thus concludes that since cyclin-dependent kinase inhibitors are classified into two distinct families that differ structurally and functionally, and since the results disclosed by Nakayama et al. indicate that members of the same family have diverse physiological functions, Applicants' disclosure of three novel cyclin-dependent kinase inhibitors , one of which is exemplified as exhibiting phenotypic effects when expressed in a plant, does not provide adequate description of the claimed genus. According to the Examiner, one skilled in the art would therefore not recognize from the disclosure that the applicant was in possession of the genus that comprises cyclin-dependent kinases that control or alter growth characteristics.

In response to the rejection of Claims 2, 5, 7-25, 27-31 and 36-57, Applicants respectfully submit the following. A showing that the written description of an invention is sufficient to inform a skilled artisan at the time the application was first filed, that applicant was in possession

of the claimed invention, may be made by many different ways. One way to show possession of the invention is by actual reduction to practice. With respect to the protein encoded by SEQ ID NO:1, methods for controlling or altering growth characteristics in a plant by overexpressing the nucleotide sequence set forth in SEQ ID NO:1, and transgenic plants which overexpress the nucleotide sequence set forth in SEQ IDNO:1, have been reduced to practice by Applicants. *See* Examples.

Reduction to practice is only *one* way to show possession of the invention. Thus, with respect to other species of cyclin-dependent kinase inhibitors encompassed by the rejected claims, Applicants may use other indicia to show possession of the invention. Thus, the written description requirement for a claimed genus may be satisfied by disclosure of "relevant, identifying characteristics, i.e., structure or other physical and/or chemical properties, by functional characteristics coupled with a known or disclosed correlation between function and structure, or by a combination of such identifying characteristics, sufficient to show the applicant was in possession of the claimed genus." Written Description Guidelines, *Fed. Reg. 66*(4), page 1106.

As presently amended, the rejected claims recite in relevant part:"[a] cyclin dependent kinase inhibitor (CKI) which interacts with CDC2a." Support for the language "which interacts with CDC2a" may be found throughout the specification. For example page 5 of the specification discloses:

> With respect to the cell cycle regulation in plants a summary of the state of the art is given below. In *Arabidopsis*, thus far only two CDK genes have been isolated, CDC2At and CDC2bAt, of which the gene products share 56% amino acid identity. Both CDKs are distinguished by several features. First, only CDC2aAt is able to complement yeast p34 CDC2/CDC28 mutants.

> Second, CDC2aAt and CDC2bAt bear different cyclin-binding motifs (PSTAIRE and PPTALRE, respectively), suggesting they may bind distinct types of cyclins. Third, although both CDC2aAt and CDC2bAt show the same spatial expression pattern, they exhibit a different cell cycle phase-specific regulation.

Page 11 of the specification indicates that "[a] CDK of the present invention is capable of inhibiting or suppressing the kinase activity of protein kinases, in particular of cylin dependent kinases. The capability of inhibiting or suppressing protein kinase activity can be determined according to methods well known in the art; see, e.g., Wang supra and the appended examples."

Example 2 demonstrates that the CKI proteins encoded by the cDNA clones LDV66, LDV39, LDV 159, associate with CDC2aAt but not with CDC2bAt. Example 4 demonstrates that FL39 and FL66 (longer clones of LV39 and LDV66, respectively, *see* specification, page 85) CKI proteins exclusively associate with CDC2aAt.

Thus, the claims as amended are fully supported by the written description as indicated in the written description guidelines, since the disclosure describes the relevant identifying characteristics recited in the claims (binding to CDC2a), i.e., functional characteristics coupled with a known or disclosed correlation between function and structure sufficient to show applicant was in possession of the claimed genus." *See Fed. Reg.* 66(4):1106.

Applicants further respectfully submit that the claims under examination are not directed to any specific cyclin-dependent kinase inhibitor but rather, to *methods of using* a cyclin-dependent kinase inhibitor (CKI) which binds CDC2a in order to control or alter growth characteristics in a plant, and to transgenic plants which express such CKIs. Part of Applicants' invention lies in the discovery that a CKI which binds CDC2a may be used to alter plant growth

characteristics. As taught by the application, *any* CKI which binds CDC2a may be used in the methods of the present invention and for producing the transgenic plants of the present invention.

With respect to the Examiner's citing Nakayama et al., Applicants submit the following. Page 12 and 82 of the present application indicate that the CKIs encoded by the isolated cDNAs of the present invention i.e., LDV39, LDV66, and LDV159, show significant homology to the human CKIs $p21^{cip1}$ and $p27^{kip1}$ in *the last 23 amino acids*. FL39 and FL66 (longer clones of LV39 and LDV66, respectively, *see* specification, page 85) CKI proteins however, are over 200 amino acids in length. Moreover, the CDK inhibitory domain of Cip/Kip CKIs as disclosed in Nakamura et al, is located at the amino terminal end of the molecules. *See* Nakamura et al., page 1021 and Figure 1. Since there are no significant homologies between the CKIs disclosed by Applicants and the CKIs disclosed by Nakayama et al. except for the carboxy terminal end, one skilled in the art would not have reasonably believed that the animal CKIs disclosed by Nakayama et al. were in any way relevant to the methods and compositions of the present invention.

The proper test for sufficiency of description in a patent application is whether the disclosure of the application relied upon "reasonably conveys to the artisan that the inventor had possession at that time of the later claimed subject matter. *In re Kaslow*, 707 F.2d 1366, 1375, 217 USPQ 1089, 1096 (Fed. Cir. 1983). Exactly how the specification allows one skilled in the art to recognize that an applicant had possession of the claimed invention is not material. *In re Smith,* 481 F.2d 910, 178 USPQ 279 (CCPA 1973). Typically, an applicant conveys that he or she is in possession of the invention by use of descriptive means such as "words, structures, figures, diagrams, formulas, etc., that set forth the claimed invention." *Lockwood v. American Airlines,* 107 F.3d 1565, 1572, 41 USPQ2d 1961, 1966 (Fed. Cir. 1997). To comply with the

description requirement, it is not necessary that the application describe the invention *ipsis verbis*. *In re Lukach*, 442 F.2d 967, 169 USPQ 795 (CCPA 1971). What is required is that an ordinarily skilled artisan recognize from the disclosure that applicants invented the subject mater of the claims, including the limitations recited therein. *In re Smith*, 481 F.2d at 915, 178 USPQ at 284.

It is respectfully submitted that the claims as presently amended are sufficiently supported by the written description provided in the specification since, based on the foregoing remarks, one skilled in the art would reasonably believe that Applicants invented the subject matter recited therein. Withdrawal of the rejection of Claims 2, 5, 7-25, 27-31 and 36-57 under 35 U.S.C. §112, first paragraph, is therefore warranted.

Claims 2, 5, 7-25, 27-31 and 36-57 have been rejected under 35 U.S.C. § 112, first paragraph as allegedly directed to non-enabled subject matter. It is the Examiner's position that the claims should be limited to a method for decreasing cyclin-dependent kinase activity in *Arabidopsis* plants which comprises introducing into a plant a nucleotide sequence of SEQ ID NO:1 encoding the homologous cyclin-dependent kinase inhibitor ICKI2 of SEQ ID NO:2, wherein said method increases the level of ICK2 in a cell, increases plant cell size in petals, leaves and stems, decreases cell number in a plant, increases leaf serration, increases the size of stomata, reduces petal size, reduces leaf venation, decreases endoreduplication and ploidy level in mature leaf cells, and reduces seed size, compared to wild type plants.

According to the Examiner, the specification does not reasonably provide enablement for methods of altering growth characteristics in plants which comprise introducing into a plant a nucleotide sequence encoding a cyclin-dependent kinase inhibitor, wherein said method modifies plant cell size, modifies cell number in a plant, alters leaf shape, alters leaf size, increases gas

exchange and photosynthesis, alters tissue or organ shape or size, alters leaf veination, facilitates the transition from the mitotic cycle to G1 arrest, alters seed size, or alters seed shape, compared to wild type plants.

Applicants traverse the rejection of Claims 2, 5, 7-25, 27-31 and 36-57 under 35 U.S.C. §112, first paragraph and respectfully submit the following. The invention of the present application lies in part, in the discovery that plants exhibit modified growth characteristics upon expression of a CKI which binds CDC2a. For example, the specification exemplifies use of the CaMV 35S promoter to drive expression of a CKI which binds CDC2a. The 35S promoter is a strong, constitutive promoter resulting in plants with many altered growth characteristics. Such results demonstrate that CKIs may be used to alter growth characteristics in plants. One skilled in the art would know that the type and timing of growth modification may be manipulated according to the choice of promoter (which may be selected from a wide range of known promoters, which selection would not involve inventive skill). For example, a skilled artisan would know that by choosing a tissue-preferred or tissue-specific promoter, more localized expression patterns (e.g., restricted to a specific part, or limited number of plant parts) would be effected. With respect to the Examiner's objection to Claim 7 encompassing methods which both increase or decrease plant cell size, objection to Claim 11 encompassing methods which both increase and decrease plant cell number, objection to Claims 15 and 39 encompassing methods and plants in which leaf size is both increased and decreased, and objection to Claims 30 and 47 encompassing methods and plants in which seed size is both increased and decreased, Applicants respectfully submit that one skilled in the art would reasonably understand from the teachings provided in the specification that opposing characteristics are obtained depending upon whether the CKI gene is up-regulated or down-regulated.

In response to the Examiner's objection that the rejected claims encompass methods and plants in which *any* growth characteristic is controlled or altered in *any* way, Applicants submit the following. The rejected claims are limited to alteration of growth characteristics due to introduction into a plant of a gene encoding a CKI which interacts with CDC2a. As further evidence that the presently claimed invention is fully enabled by the specification, submitted herewith as Exhibit A is an article by DeVeylder et al. (2001) "Functional Analysis of Cyclin - Dependent Kinase Inhibitors of Arabidopsis" *The Plant Cell, 13*:1653-1667. The DeVeylder article describes additional CKIs which interact with CDC2a, isolated following the methods taught in the present application. For example, a CKI designated KRP3 or CKI3 was demonstrated to interact with CDKA1 (CDC2a) but not CDKB1;1 (CDC2b). *See* Table 2. Binding of CDKA1 was confirmed by *in vitro* assay. *See* page 1656, first full paragraph. Expression of CKI3 in a transgenic plant results in the same leaf phenotypes as taught by the present application.

Also submitted herewith as Exhibit B is an article by Zhou et al. (2002) "Plant CDK inhibitors: studies of interactions with cell cycle regulators in the yeast two hybrid system and functional comparison in transgenic *Arabidopsis* plants" *Plant Cell Rep. 20*:967-975. The Zhou et al. article confirms results reported by DeVeylder et al.(Exhibit A). For example, all CKI proteins which interact with CDC2a and that were over expressed in plants under control of the CaMV 35S promoter showed the same phenotype: altered leaf and flower morphology, reduced nuclear DNA content (endoreduplication was affected). Thus, the Zhou et al. article also provides further evidence that following the teachings of the present application, additional CKIs which interact with CDC2a have been isolated and used in the methods of the present application to alter growth characteristics of plants.

On page 8 of the Office Action, the Examiner cites Riou-Khamiichi et al. (March 5, 1999) *Science* 283:1541-1544 and Cockcroft et al. (June 1, 2000) *Nature 405*:575-579 as evidence that altering growth characteristics in a plant by expressing a heterologous cell cycle protein is highly unpredictable. It is respectfully submitted that both Riou-Khamiichi et al. and Cockcroft et al. concern expression of a cyclin D gene. Thus, at most, the two references teach that expression of a cyclin D gene in a plant gives rise to a range of phenotypes. There is no evidence provided by either reference that expression of a CKI would behave similarly. Moreover, faced with a population of plants displaying a range of phenotypes, one skilled in the art would know to select plants displaying desirable characteristics.

In order to be considered enabling, the specification must teach a skilled artisan how to make and use the full scope of the claimed invention without "undue experimentation." *Genentech Inc. v. Novo Nordisk, A/S,* 108 F.3d 1361, 1365, 42 USPQ2d 1001, 1004 (Fed. Cir. 1997); *In re Wright*, 999 F. 2d 1557, 1561, 27 USPQ2d 1510, 1513 (Fed. Cir. 1993). In performing the analysis, the key word is "undue", not "experimentation." *In re Angstadt*, 537 F.2d 498, 504, 190 USPQ 214, 219 (CCPA 1976). The question of whether the claims of a patent are sufficiently enabled by a disclosure in a specification is determined as of the date the patent application was first filed. *Hybritech, Inc. v. Monoclonal Antibodies, Inc.*, 802 F.2d 1367, 1384, 231 USPQ 81, 94 (Fed. Cir. 1986). Whether undue experimentation would have been required at the time the application was originally filed is not a single, simple factual determination, but is a conclusion reached by weighing many factual considerations. *In re Wands*, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988). The test is not merely quantitative, as a considerable amount of experimentation is permissible, if it is merely routine (such as routine screening), or if the specification in question provides a reasonable amount of

guidance with respect to the direction in which the experimentation should proceed. *In re Wands*, 858 F.2d at 737, 8 USPQ2d at 1404.

The specification provides ample direction with respect to isolating CKI genes which interact with CDC2a (*see* specification, pages 10-21, and examples 1-4), constructing vectors comprising such genes (*see* specification, page 21-22), and targeting phenotypic effects to specific plant parts and plant tissues by using an appropriate promoter which functions in plants (*see* specification, page 22-24, 64-69, and examples 10 and 11). Well known methods of transformation of different plant species are also discussed in the specification (see specification, pages 32-34, 69-70). Applicants respectfully submit that although *some* experimentation may be necessary in order to practice the claimed invention, such experimentation involves routine screening and selection of plant phenotype.

In view of the amendments to the claims and the remarks hereinabove, Applicants respectfully request withdrawal of the rejection of Claims 2, 5, 7-25, 27-31 and 36-57 under 35 U.S.C. 112, first paragraph.

Claims 2, 5, 7-25, 27-29, 36-39 and 42-57 have been rejected under 35 U.S.C. 112, second paragraph, as allegedly indefinite for failing to particularly point out and distinctly claim the subject matter of the invention. Claims 2, 5, 7, 11, 14, 17, 18, 19, 21, 25, 27, 30, and 31 are objected to in particular for allegedly omitting essential steps. As presently amended, Claims 2, 5, 7, 11,14, 17, 18, 19, 21, 25, 27, 30, and 31 recite all essential steps specifically indicated as missing by the Examiner. In addition, Claim 5 has been amended to recite a basis for comparison for "increasing the level of cyclin-dependent kinase inhibitor." Similarly, Claim 17 presently recites a basis for comparison of "increasing stomata size in a plant." Claim 18 is

presently amended to recite a basis for comparison for "increasing gas exchange and photosynthesis."

Claim 27 is specifically objected to due to its recitation of "facilitating." As presently amended, Claim 27 no longer recites "facilitating" and recites in relevant part: "[a] method of promoting the transition from the mitotic cycle to G1 arrest in a plant cell..." Claim 36 is specifically objected to due to its recitation of "an essentially derived variety thereof." As presently amended, Claim 36 recites in relevant part: "a transgenic plant, a variety derived thereof with essentially the same characteristics, ...". Support for the amendment may be found throughout the specification, e.g., page 37, lines 13-19.

Claims 46, 48, 49, and 50 have been objected to in particular as lacking a basis for comparison. As presently amended, Claim 46 recites in relevant part "decreased ploidy level relative to corresponding wild type plants." Claim 48 has been amended to recite in relevant part "the total cell number of the plant is decreased relative to corresponding wild type plants." Claim 49 presently recites "comprising cells of increased size relative to corresponding wild type plants." Claim 50 as presently amended recites in relevant part: "having increased photosynthetic capability relative to corresponding wild type plants."

In view of Applicants' amendments to the claims, withdrawal of the rejection of Claims 2, 5, 7-25, 27-29, 36-39 and 42-57 under 35 U.S.C.§ 112, second paragraph is respectfully requested.

Claims 56 and 57 have been rejected under 35 U.S.C. §101 as allegedly directed to non-statutory subject matter. As presently amended, Claim 56 recites "[h]arvestable parts or propagation material from the transgenic plant of Claim 36, comprising the nucleotide sequence encoding a CKI which interacts with CDC2a, that was introduced into the parent plant." Claim

57 presently recites "[c]ut flowers from the transgenic plant of claim 36, comprising the nucleotide sequence encoding a CKI which interacts with CDC2a, that was introduced into the parent plant." In view of the amendment to the claims, Applicants respectfully request withdrawal of the rejection of Claims 56 and 57 under 35 U.S.C. §101.

Claims 2, 5, 7 -25, 27-31, 36-51 and 56-57 have been rejected under 35 U.S.C.§ 102(a) as allegedly anticipated by or, in the alternative, under 35 U.S.C. §103(a) as obvious over WO 99/64599 which published on December 16, 1999.

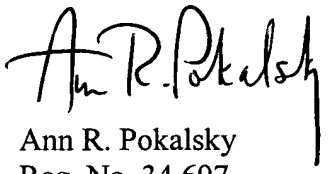
Applicants traverse the rejection under either or both statutory sections and respectfully submit that the rejection is in error for the following reasons. Applicants have claimed benefit of priority from earlier filed applications under both 35 U.S.C. § 120 and 35 U.S.C. § 119. Such claim of priority was duly filed with the transmittal papers of the above-identified application as well as part of the combined inventors' declaration/power of attorney. Indeed, the priority information is indicated on the filing receipt for the above-identified application. Thus, the present application, U.S. Serial No. 09/574,735, filed in the U.S. Patent and Trademark Office on May 18, 2000 is a continuation-in-part application to U.S. Serial No. 09/526,597 (presently pending) which was filed in the U.S. Patent and Trademark Office on March 16, 2000. U.S. Serial No. 09/526,597 was filed as a national phase application (section 371 application) to PCT/EP98/05895 having an International Filing date of September 16, 1998 and an International Publication date of March 25, 1999 (both of which dates precede the international publication date of WO 99/64599). PCT/EP98/05895 claims priority from two European patent applications, EP 97 204 111.5, filed December 24, 1997 and EP 97 202 838.5, filed September 16, 1997. As the priority dates of September 16, 1998, December 24, 1997 and September 16, 1997 are all prior to the publication date of WO 99/64599 (December 16, 1999), the invention disclosed in

the present application is antedated with respect to WO 99/64599. For the Examiner's convenience, a copy of PCT/EP98/05895 (WO 99/14331) is enclosed herewith.

Due to Applicants' previously and timely filed claim of priority under 35 U.S.C §119 and §120, withdrawal of the rejection of Claims 2, 5, 7 -25, 27-31, 36-51 and 56-57 under 35 U.S.C.§ 102(a) and/or 35 U.S.C. §103(a) is warranted.

In view of the foregoing remarks and amendments, it is firmly believed that the present application is in condition for allowance, which action is earnestly solicited.

Respectfully submitted,



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VERSION WITH MARKINGS TO SHOW CHANGES MADE

2. (Amended) A method for controlling or altering growth characteristics in a plant, [which comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a regulatory sequence which controls expression of the cyclin-dependent kinase inhibitor;

(ii) expressing said nucleic acid molecule; and

(iii) regenerating a plant therefrom, which plant has altered growth characteristics.

5. (Amended) A method for increasing the level of cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, in a plant cell [which comprises] relative to corresponding cells of a wild type plant, said method comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor under the control of a promoter which functions in plants; and

(ii) expressing said nucleic acid molecule in said plant cell, thereby increasing the level of cyclin-dependent kinase inhibitor in said plant cell.

7. (Amended) A method for modifying plant cell size, [which] said method [comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of promoter which functions in plants; and

(ii) expressing said nucleic acid molecule in said plant cell, thereby modifying plant cell size.

11. (Amended) A method for modifying cell number in a plant, [which comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a promoter which functions in plants;

(ii) expressing said nucleic acid molecule in said plant cell; and

(iii) regenerating a plant from said plant cell, wherein said plant has [with] modified cell number.

12. (Amended) The method according to claim [6,] 7, 8, 9 or 10 wherein plant cell size is increased.

14. (Amended) A method of altering leaf shape in a plant, [which comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a promoter which functions in plants;

(ii) expressing said nucleic acid molecule in said plant cell; and

(iii) regenerating a plant [therefrom] from said plant cell, said plant having altered leaf shape.

15. (Amended) A method of altering leaf size in a plant, [which comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a promoter which functions in plants;

(ii) expressing said nucleic acid molecule in said plant cell; and

(iii) regenerating a plant [therefrom having] from said plant cell, wherein said plant has altered leaf size.

17. (Amended) A method of increasing stomata size of a plant, [which comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a promoter which functions in plants;

(ii) expressing said nucleic acid molecule in said plant cell; and

(iii) regenerating a plant [therefrom] from said plant cell, said plant having increased stomata size relative to corresponding wild type plants.

18. (Amended) A method of increasing gas exchange and photosynthesis in a plant, [which comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a promoter which functions in plants;

(ii) expressing said nucleic acid molecule in said plant cell; and

(iii) regenerating a plant [therefrom] from said plant cell, said plant having increased gas exchange and photosynthesis relative to corresponding wild type plants.

19. (Amended) A method of altering tissue or organ shape in a plant, [which comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a promoter which functions in plants;

(ii) expressing said nucleic acid molecule in said plant cell; and

(iii) regenerating a plant [therefrom having] from said plant cell, wherein said plant has flowers with altered petal shape.

21. (Amended) A method of altering tissue or organ size in a plant, [which comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a promoter which functions in plants;

(ii) expressing said nucleic acid molecule in the plant cell; and

(iii) regenerating a plant [therefrom having] from said plant cell, wherein said plant has flowers with altered petal size.

25. (Amended) A method of altering venation pattern in a plant leaf, [which comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a promoter which functions in plants;

(ii) expressing said nucleic acid molecule in the plant cell; and

(iii) regenerating a plant [therefrom having] from said plant cell, wherein said plant has leaves with an altered venation pattern.

27. (Amended) A method of [facilitating] promoting the transition from the mitotic cycle to G1 arrest in a plant cell, [which comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a promoter which functions in plants; and

(ii) expressing aid nucleic acid molecule in the plant cell.

28. (Amended) The method of claim [26 or] 27 wherein said facilitating the transition from the mitotic cycle to G1 arrest in a plant cell results in a decrease in endoreduplication in the plant cell.

29. (Amended) The method of claim [26 or] 27 wherein said facilitating the transition from the mitotic cycle to G1 arrest in a plant cell results in a decrease in ploidy level in the plant cell.

30. (Amended) A method of altering plant seed size, [which comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a promoter which functions in plants;

(ii) expressing said nucleic acid molecule in the plant cell; and

(iii) regenerating a plant [therefrom having] from said plant cell, wherein said plant has decreased seed size [compared] relative to corresponding wild type plants.

31. (Amended) A method of altering plant seed shape, [which comprises] comprising the steps of:

(i) introducing into a plant cell a nucleic acid molecule encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a promoter which functions in plants;

(ii) expressing said nucleic acid molecule in the plant cell; and

(iii) regenerating a plant [therefrom] from said plant cell, said plant having decreased seed shape [compared] relative to corresponding wild type plants.

36. (Amended) A transgenic plant, [an] a variety derived thereof with essentially [derived variety thereof,] the same characteristics, a plant part, or plant cell which comprises a nucleotide sequence encoding a cyclin-dependent kinase inhibitor (CKI) which interacts with CDC2a, under the control of a promoter which functions in plants wherein said nucleotide sequence encoding a cyclin-dependent kinase inhibitor is heterologous to the genome of the transgenic plant, or is homologous but additional to the genome of the transgenic plant or has been introduced into the transgenic plant, plant part or plant cell by recombinant DNA means.

46. (Amended) The transgenic plant of claim 37, wherein the cells have [an] a decreased ploidy level relative to corresponding wild type plants.

48. (Amended) The transgenic plant of claim 36, wherein the total cell number of the plant is decreased relative to corresponding wild type plants.

49. (Amended) The transgenic plant of claim 36, comprising cells of increased size relative to corresponding wild type plants.

50. (Amended) The transgenic plant of claim 36, comprising leaves with increased stomata size relative to corresponding wild type plants.

51. (Amended) The transgenic plant of claim 36 having increased photosynthetic capacity relative to corresponding wild type plants.

52. (Amended) The method of claims [1-11, 13-27, or 30-36] 2, 5, 7, 11, 14, 15, 17-19, 21, 25, 27, 30 or 31, wherein the CKI comprises the amino acid sequence as set forth in [any one of] SEQ ID NO: 2 [Nos:2, 4, or 6].

53. (Amended) The method of claims 2, 5, [13-27] 7-11, 13-25, 27, 30, and 31, [30-35] wherein the nucleic acid molecule comprises the nucleotide sequence as set forth in [any one of] SEQ ID NO:1[, 3, or 5].

54. (Amended) The method of [of] claims [1-4, 13-27, 30-36] 2, 5, 7-11, 13-25, 27, 30 and 31, wherein the CKI comprises the consensus amino acid sequence as set forth in any one of SEQ ID NO:34, SEQ ID NO:35, SEQ ID NO:36, SEQ ID NO:37, SEQ ID NO:38 or SEQ ID NO:39.

56. (Amended) Harvestable parts or propagation material from the transgenic plant of claim 36, comprising the (CKI) which interacts with CDC2a, that was introduced into the parent plant.

57. (Amended) Cut flowers from the transgenic plant of claim 36, comprising the (CKI) which interacts with CDC2a, that was introduced into the parent plant.